```
FILE 'HOME' ENTERED AT 12:12:05 ON 11 NOV 2009

=> FIL REGISTRY
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                       0.22       0.22

FILE 'REGISTRY' ENTERED AT 12:12:35 ON 11 NOV 2009
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STRUCTURE FILE UPDATES:   10 NOV 2009  HIGHEST RN 1192107-32-6
DICTIONARY FILE UPDATES:  10 NOV 2009  HIGHEST RN 1192107-32-6

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH June 26, 2009.

  Please note that search-term pricing does apply when
  conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and
predicted properties as well as tags indicating availability of
experimental property data in the original document.  For information
on property searching in REGISTRY, refer to:

http://www.cas.org/support/stngen/stndoc/properties.html

=> E "ARSENIC"/CN 25
E1            1     ARSENIAN STIBIOLUZONITE/CN
E2            1     ARSENIAN STRONTIAN GAMAGARITE/CN
E3            1 --> ARSENIC/CN
E4            1     ARSENIC (+3 OXIDATION STATE) METHYLTRANSFERASE (ALCANIVORAX
BORKUMENSIS STRAIN SK2)/CN
E5            1     ARSENIC (AS1+)/CN
E6            1     ARSENIC (AS2)/CN
E7            1     ARSENIC (AS3)/CN
E8            1     ARSENIC (AS4)/CN
E9            1     ARSENIC (AS41+)/CN
E10           1     ARSENIC (III)/CN
E11           1     ARSENIC (III) METHYLTRANSFERASE (OIKOPLEURA DIOICA CLONE
BACOIKO007-10XI11)/CN
E12           1     ARSENIC (V) OXIDE/CN
E13           1     ARSENIC 0-0.1, CARBON 0.2, CHROMIUM 0.4-0.6, COPPER 0-0.2, IRON
96-98, MANGANESE 0.6-1, MOLYBDENUM 0.2, NICKEL 0.4-0.8, SILICON 0.2-0.4, VANADIUM
0-0.1/CN
E14           1     ARSENIC 0-0.1, COPPER 99.9-100 (ATOMIC)/CN
E15           1     ARSENIC 0-0.2, CADMIUM 100/CN
E16           1     ARSENIC 0-1, CARBON 7, IRON 79-80, PHOSPHORUS 13 (ATOMIC)/CN
E17           1     ARSENIC 0-1, GERMANIUM 45-55, TELLURIUM 45-55 (ATOMIC)/CN
E18           1     ARSENIC 0-1, NICKEL 99-100 (ATOMIC)/CN
E19           1     ARSENIC 0-1.5, SILVER 98.5-100 (ATOMIC)/CN
E20           1     ARSENIC 0-2, LEAD 98-100/CN
E21           1     ARSENIC 0-2,COPPER 90.5-92.5,INDIUM 7.5 (ATOMIC)/CN
E22           1     ARSENIC 0-21, TIN 79-100 (ATOMIC)/CN
E23           1     ARSENIC 0-22, LEAD 78-100 (ATOMIC)/CN
E24           1     ARSENIC 0-25, BORON 0-25, PALLADIUM 75 (ATOMIC)/CN
```
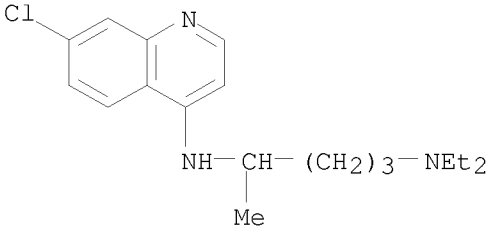

```
E25          1     ARSENIC 0-3.1, SILVER 96.9-100 (ATOMIC)/CN

=> S E3
L1           1 ARSENIC/CN

=> DIS L1 1 IDE
THE ESTIMATED COST FOR THIS REQUEST IS 2.05 U.S. DOLLARS
DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N:Y

L1   ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2009 ACS on STN
RN   7440-38-2  REGISTRY
ED   Entered STN:  16 Nov 1984
CN   Arsenic  (CA INDEX NAME)
OTHER NAMES:
CN   Arsenic black
CN   Arsenic-75
DR   55624-62-9, 39277-51-5
MF   As
CI   COM
LC   STN Files:   ADISNEWS, AGRICOLA, ANABSTR, AQUIRE, BIOSIS, BIOTECHNO, CA,
       CABA, CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMLIST, CIN, CSCHEM, CSNB,
       DDFU, DETHERM*, DRUGU, EMBASE, ENCOMPLIT, ENCOMPLIT2, ENCOMPPAT,
       ENCOMPPAT2, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*,
       MSDS-OHS, PIRA, PROMT, PS, RTECS*, TOXCENTER, TULSA, ULIDAT, USPAT2,
       USPATFULL, VETU
         (*File contains numerically searchable property data)
     Other Sources:   DSL**, EINECS**, TSCA**
         (**Enter CHEMLIST File for up-to-date regulatory information)


As



**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

           99468 REFERENCES IN FILE CA (1907 TO DATE)
            3642 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
           99738 REFERENCES IN FILE CAPLUS (1907 TO DATE)


=> E "ARSENIC"/CN 25
E1           1     ARSENIAN STIBIOLUZONITE/CN
E2           1     ARSENIAN STRONTIAN GAMAGARITE/CN
E3           1 --> ARSENIC/CN
E4           1     ARSENIC (+3 OXIDATION STATE) METHYLTRANSFERASE (ALCANIVORAX
BORKUMENSIS STRAIN SK2)/CN
E5           1     ARSENIC (AS1+)/CN
E6           1     ARSENIC (AS2)/CN
E7           1     ARSENIC (AS3)/CN
E8           1     ARSENIC (AS4)/CN
E9           1     ARSENIC (AS41+)/CN
E10          1     ARSENIC (III)/CN
E11          1     ARSENIC (III) METHYLTRANSFERASE (OIKOPLEURA DIOICA CLONE
BACOIKO007-10XI11)/CN
E12          1     ARSENIC (V) OXIDE/CN
E13          1     ARSENIC 0-0.1, CARBON 0.2, CHROMIUM 0.4-0.6, COPPER 0-0.2, IRON
96-98, MANGANESE 0.6-1, MOLYBDENUM 0.2, NICKEL 0.4-0.8, SILICON 0.2-0.4, VANADIUM
0-0.1/CN
E14          1     ARSENIC 0-0.1, COPPER 99.9-100 (ATOMIC)/CN
```

```
E15           1     ARSENIC 0-0.2, CADMIUM 100/CN
E16           1     ARSENIC 0-1, CARBON 7, IRON 79-80, PHOSPHORUS 13 (ATOMIC)/CN
E17           1     ARSENIC 0-1, GERMANIUM 45-55, TELLURIUM 45-55 (ATOMIC)/CN
E18           1     ARSENIC 0-1, NICKEL 99-100 (ATOMIC)/CN
E19           1     ARSENIC 0-1.5, SILVER 98.5-100 (ATOMIC)/CN
E20           1     ARSENIC 0-2, LEAD 98-100/CN
E21           1     ARSENIC 0-2,COPPER 90.5-92.5,INDIUM 7.5 (ATOMIC)/CN
E22           1     ARSENIC 0-21, TIN 79-100 (ATOMIC)/CN
E23           1     ARSENIC 0-22, LEAD 78-100 (ATOMIC)/CN
E24           1     ARSENIC 0-25, BORON 0-25, PALLADIUM 75 (ATOMIC)/CN
E25           1     ARSENIC 0-3.1, SILVER 96.9-100 (ATOMIC)/CN

=> E "CHLOROQUINE"/CN 25
E1            1     CHLOROQUANIL/CN
E2            1     CHLOROQUIN DIPHOSPHATE/CN
E3            1 --> CHLOROQUINE/CN
E4            1     CHLOROQUINE 2,5-DIHYDROXYBENZOATE/CN
E5            1     CHLOROQUINE ARTESUNATE/CN
E6            1     CHLOROQUINE ASCORBATE/CN
E7            1     CHLOROQUINE CHONDROITIN SULFATE/CN
E8            1     CHLOROQUINE DIASCORBATE/CN
E9            1     CHLOROQUINE DIHYDROCHLORIDE/CN
E10           1     CHLOROQUINE DIHYDROGEN PHOSPHATE (1:2)/CN
E11           1     CHLOROQUINE DIOROTATE/CN
E12           1     CHLOROQUINE DIPHOSPHATE/CN
E13           1     CHLOROQUINE DIPHOSPHATE MONOHYDRATE/CN
E14           1     CHLOROQUINE HYDROCHLORIDE/CN
E15           1     CHLOROQUINE MUSTARD/CN
E16           1     CHLOROQUINE N-OXIDE/CN
E17           1     CHLOROQUINE PHOSPHATE/CN
E18           1     CHLOROQUINE PHOSPHATE-PROGUANIL HYDROCHLORIDE MIXT./CN
E19           1     CHLOROQUINE RESISTANCE MARKER PROTEIN (PLASMODIUM FALCIPARUM
STRAIN 3D7 GENE PF14-0463)/CN
E20           1     CHLOROQUINE RESISTANCE PROTEIN (PLASMODIUM FALCIPARUM STRAIN 7G8
GENE CG2)/CN
E21           1     CHLOROQUINE RESISTANCE PROTEIN (PLASMODIUM FALCIPARUM STRAIN HB3
GENE CG2)/CN
E22           1     CHLOROQUINE RESISTANCE TRANSPORTER (CRT)-LIKE PROTEIN
(PLASMODIUM CHABAUDI CLONE AS GENE CG10)/CN
E23           1     CHLOROQUINE RESISTANCE TRANSPORTER (PLASMODIUM FALCIPARUM CLONE
106/1 GENE CRT)/CN
E24           1     CHLOROQUINE RESISTANCE TRANSPORTER (PLASMODIUM FALCIPARUM CLONE
7G8 GENE CRT)/CN
E25           1     CHLOROQUINE RESISTANCE TRANSPORTER (PLASMODIUM FALCIPARUM CLONE
DIV30 GENE CRT)/CN

=> S E3
L2            1 CHLOROQUINE/CN

=> DIS L2 1 IDE
THE ESTIMATED COST FOR THIS REQUEST IS 2.05 U.S. DOLLARS
DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N:Y

L2   ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2009 ACS on STN
RN   54-05-7  REGISTRY
ED   Entered STN:  16 Nov 1984
CN   1,4-Pentanediamine, N4-(7-chloro-4-quinolinyl)-N1,N1-diethyl-  (CA INDEX
     NAME)
OTHER CA INDEX NAMES:
CN   Quinoline, 7-chloro-4-[[4-(diethylamino)-1-methylbutyl]amino]- (8CI)
OTHER NAMES:
```

CN (±)-Chloroquine
CN 7-Chloro-4-[[4-(diethylamino)-1-methylbutyl]amino]quinoline
CN Aralen
CN Artrichin
CN Bipiquin
CN Capquin
CN Chloraquine
CN Chlorochin
CN Chloroquine
CN NSC 187208
CN Reumachlor
CN Ronaquine
CN RP 3377
CN ST 121
CN ST 121 (pharmaceutical)
DR 56598-66-4
MF C18 H26 Cl N3
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOSIS, BIOTECHNO, CA, CABA, CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DRUGU, EMBASE, HSDB*, IFICDB, IFIPAT, IFIUDB, IMSPRODUCT, IPA, MEDLINE, MRCK*, PIRA, PROMT, PS, RTECS*, SPECINFO, TOXCENTER, USAN, USPAT2, USPATFULL, USPATOLD, VETU
(*File contains numerically searchable property data)
Other Sources: EINECS**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)




**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

5751 REFERENCES IN FILE CA (1907 TO DATE)
94 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
5768 REFERENCES IN FILE CAPLUS (1907 TO DATE)

=> index bioscience
FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED

| COST IN U.S. DOLLARS | SINCE FILE ENTRY | TOTAL SESSION |
|---|---|---|
| FULL ESTIMATED COST | 16.24 | 16.46 |

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE, AQUASCI, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CAPLUS, CEABA-VTB, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB, DRUGMONOG2, DRUGU, EMBAL, EMBASE, ...' ENTERED AT 12:14:15 ON 11 NOV 2009

68 FILES IN THE FILE LIST IN STNINDEX

Enter SET DETAIL ON to see search term postings or to view

search error messages that display as 0* with SET DETAIL OFF.

=> b reg
COST IN U.S. DOLLARS | SINCE FILE ENTRY | TOTAL SESSION
FULL ESTIMATED COST | 0.68 | 17.14

FILE 'REGISTRY' ENTERED AT 12:14:19 ON 11 NOV 2009
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STRUCTURE FILE UPDATES: 10 NOV 2009 HIGHEST RN 1192107-32-6
DICTIONARY FILE UPDATES: 10 NOV 2009 HIGHEST RN 1192107-32-6

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH June 26, 2009.

Please note that search-term pricing does apply when
conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and
predicted properties as well as tags indicating availability of
experimental property data in the original document. For information
on property searching in REGISTRY, refer to:

http://www.cas.org/support/stngen/stndoc/properties.html

=> sel L2 chem
E1 THROUGH E17 ASSIGNED

=> index bioscience
FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED
COST IN U.S. DOLLARS | SINCE FILE ENTRY | TOTAL SESSION
FULL ESTIMATED COST | 0.86 | 18.00

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE,
AQUASCI, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CAPLUS,
CEABA-VTB, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB,
DRUGMONOG2, DRUGU, EMBAL, EMBASE, ...' ENTERED AT 12:14:33 ON 11 NOV 2009

68 FILES IN THE FILE LIST IN STNINDEX

Enter SET DETAIL ON to see search term postings or to view
search error messages that display as 0* with SET DETAIL OFF.

=> s e1-17 or arsen### or retin### (s) lysosom##
646 FILE ADISCTI
42 FILE ADISINSIGHT
533 FILE ADISNEWS
4811 FILE AGRICOLA
6579 FILE ANABSTR
1312 FILE ANTE
3701 FILE AQUALINE
3461 FILE AQUASCI
2140 FILE BIOENG

```
    42969   FILE BIOSIS
      835   FILE BIOTECHABS
      835   FILE BIOTECHDS
     4978   FILE BIOTECHNO
13 FILES SEARCHED...
    19184   FILE CABA
   378054   FILE CAPLUS
     1332   FILE CEABA-VTB
     1196   FILE CIN
     2724   FILE CONFSCI
      623   FILE CROPB
      771   FILE CROPU
     4248   FILE DDFB
     8280   FILE DDFU
     2533   FILE DGENE
23 FILES SEARCHED...
     6276   FILE DISSABS
     4248   FILE DRUGB
      921   FILE DRUGMONOG2
     9040   FILE DRUGU
      233   FILE EMBAL
    43870   FILE EMBASE
    12381   FILE ESBIOBASE
        3   FILE FOMAD
        2   FILE FOREGE
     1241   FILE FROSTI
     1954   FILE FSTA
34 FILES SEARCHED...
    49738   FILE GENBANK
     1585   FILE HEALSAFE
    23987   FILE IFIPAT
       69   FILE IMSDRUGNEWS
      267   FILE IMSPRODUCT
       39   FILE IMSRESEARCH
       93   FILE KOSMET
    11557   FILE LIFESCI
    35091   FILE MEDLINE
    11652   FILE NTIS
       36   FILE NUTRACEUT
      610   FILE OCEAN
    43096   FILE PASCAL
47 FILES SEARCHED...
       18   FILE PCTGEN
       42   FILE PHAR
       95   FILE PHARMAML
      708   FILE PHIN
    43225   FILE PROMT
      243   FILE PROUSDDR
        2   FILE PS
      113   FILE RDISCLOSURE
    52238   FILE SCISEARCH
        6   FILE SYNTHLINE
    83726   FILE TOXCENTER
      579   FILE USGENE
59 FILES SEARCHED...
   111400   FILE USPATFULL
    13520   FILE USPATOLD
    24709   FILE USPAT2
      796   FILE VETB
      263   FILE VETU
64 FILES SEARCHED...
```

```
       5012   FILE WATER
      38379   FILE WPIDS
         63   FILE WPIFV
      38379   FILE WPINDEX

  68 FILES HAVE ONE OR MORE ANSWERS,   68 FILES SEARCHED IN STNINDEX

L3   QUE ("(±)-CHLOROQUINE"/BI OR ARALEN/BI OR ARTRICHIN/BI OR BIPIQUIN/BI O
         R CAPQUIN/BI OR CHLORAQUINE/BI OR CHLOROCHIN/BI OR CHLOROQUINE/BI OR "
         NSC 187208"/BI OR REUMACHLOR/BI OR RONAQUINE/BI OR "RP 3377"/BI OR "ST
          121 (PHARMACEUTICAL)"/BI OR "ST 121"/BI OR 54-05-7/BI OR 56598-66-4/B
         I OR "7-CHLORO-4-((4-(DIETHYLAMINO)-1-METHYLBUTYL)AMINO)QUINOLINE"/BI)
          OR ARSEN### OR RETIN### (S) LYSOSOM##

=> s L3 (s) (disrupt#### or destabiliz### or permea###### or degrad#####)
           1   FILE ADISCTI
           4   FILE ADISINSIGHT
           5   FILE ADISNEWS
         150   FILE AGRICOLA
          40   FILE ANABSTR
          10   FILE ANTE
         103   FILE AQUALINE
         121   FILE AQUASCI
         193   FILE BIOENG
        1125   FILE BIOSIS
         106   FILE BIOTECHABS
         106   FILE BIOTECHDS
         664   FILE BIOTECHNO
         484   FILE CABA
        2676   FILE CAPLUS
          46   FILE CEABA-VTB
           7   FILE CIN
          18   FILE CONFSCI
  18 FILES SEARCHED...
           8   FILE CROPB
          26   FILE CROPU
          73   FILE DDFB
         263   FILE DDFU
         136   FILE DGENE
         235   FILE DISSABS
          73   FILE DRUGB
         415   FILE DRUGU
  27 FILES SEARCHED...
           7   FILE EMBAL
         921   FILE EMBASE
         956   FILE ESBIOBASE
          17   FILE FROSTI
          40   FILE FSTA
         983   FILE GENBANK
          12   FILE HEALSAFE
         182   FILE IFIPAT
           1   FILE IMSDRUGNEWS
           1   FILE IMSRESEARCH
           3   FILE KOSMET
         796   FILE LIFESCI
         985   FILE MEDLINE
         174   FILE NTIS
          19   FILE OCEAN
         883   FILE PASCAL
  47 FILES SEARCHED...
           2   FILE PHIN
```

```
       230   FILE PROMT
         5   FILE PROUSDDR
         1   FILE RDISCLOSURE
       800   FILE SCISEARCH
       897   FILE TOXCENTER
      2954   FILE USPATFULL
       200   FILE USPATOLD
  61 FILES SEARCHED...
       514   FILE USPAT2
         5   FILE VETU
       177   FILE WATER
       249   FILE WPIDS
         1   FILE WPIFV
       249   FILE WPINDEX

  56 FILES HAVE ONE OR MORE ANSWERS,   68 FILES SEARCHED IN STNINDEX

L4   QUE L3 (S) (DISRUPT#### OR DESTABILIZ### OR PERMEA###### OR DEGRAD#####)

=> s (e1-17 or arsen### or retin###) (3a) lysosom##
         2   FILE ADISNEWS
        17   FILE AGRICOLA
         1   FILE ANABSTR
         3   FILE AQUASCI
         7   FILE BIOENG
       506   FILE BIOSIS
         8   FILE BIOTECHABS
         8   FILE BIOTECHDS
        98   FILE BIOTECHNO
        40   FILE CABA
       607   FILE CAPLUS
         5   FILE CONFSCI
        19   FILE DDFB
        22   FILE DDFU
       114   FILE DGENE
  23 FILES SEARCHED...
        17   FILE DISSABS
        19   FILE DRUGB
        42   FILE DRUGU
         2   FILE EMBAL
       348   FILE EMBASE
       128   FILE ESBIOBASE
        48   FILE GENBANK
        10   FILE IFIPAT
         1   FILE KOSMET
        78   FILE LIFESCI
       378   FILE MEDLINE
         1   FILE NTIS
        91   FILE PASCAL
  47 FILES SEARCHED...
         1   FILE PHIN
         2   FILE PROMT
       242   FILE SCISEARCH
       271   FILE TOXCENTER
       136   FILE USPATFULL
        23   FILE USPAT2
         1   FILE VETB
         4   FILE WPIDS
  67 FILES SEARCHED...
         4   FILE WPINDEX
```

```
  37 FILES HAVE ONE OR MORE ANSWERS,   68 FILES SEARCHED IN STNINDEX

L5   QUE (E1-17 OR ARSEN### OR RETIN###) (3A) LYSOSOM##

=> s L5 (s) (disrupt#### or destabiliz### or permea###### or degrad#####)
          14   FILE BIOSIS
           1   FILE BIOTECHABS
           1   FILE BIOTECHDS
           1   FILE BIOTECHNO
           1   FILE CABA
          19   FILE CAPLUS
           2   FILE DDFU
          22   FILE DGENE
           2   FILE DRUGU
  28 FILES SEARCHED...
          13   FILE EMBASE
<------User Break------>

=> s L5 (5a) (disrupt#### or destabiliz### or permea###### or degrad#####)
          12   FILE BIOSIS
           1   FILE BIOTECHABS
           1   FILE BIOTECHDS
          14   FILE CAPLUS
           2   FILE DDFU
           2   FILE DRUGU
          10   FILE EMBASE
           4   FILE ESBIOBASE
          24   FILE GENBANK
  37 FILES SEARCHED...
           9   FILE MEDLINE
           5   FILE PASCAL
           9   FILE SCISEARCH
           9   FILE TOXCENTER
           1   FILE USPATFULL
           1   FILE WPIDS
           1   FILE WPINDEX

  16 FILES HAVE ONE OR MORE ANSWERS,   68 FILES SEARCHED IN STNINDEX

L6   QUE L5 (5A) (DISRUPT#### OR DESTABILIZ### OR PERMEA###### OR DEGRAD#####)

=> d rank
F1           24   GENBANK
F2           14   CAPLUS
F3           12   BIOSIS
F4           10   EMBASE
F5            9   MEDLINE
F6            9   SCISEARCH
F7            9   TOXCENTER
F8            5   PASCAL
F9            4   ESBIOBASE
F10           2   DDFU
F11           2   DRUGU
F12           1   BIOTECHABS
F13           1   BIOTECHDS
F14           1   USPATFULL
F15           1   WPIDS
F16           1   WPINDEX


=> fil f2-f7, f9-f16
```

```
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                      12.24      30.24

FILE 'CAPLUS' ENTERED AT 12:25:25 ON 11 NOV 2009
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FILE 'MEDLINE' ENTERED AT 12:25:25 ON 11 NOV 2009

FILE 'SCISEARCH' ENTERED AT 12:25:25 ON 11 NOV 2009
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FILE 'TOXCENTER' ENTERED AT 12:25:25 ON 11 NOV 2009
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FILE 'ESBIOBASE' ENTERED AT 12:25:25 ON 11 NOV 2009
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FILE 'DDFU' ACCESS NOT AUTHORIZED

FILE 'DRUGU' ENTERED AT 12:25:25 ON 11 NOV 2009
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FILE 'BIOTECHABS' ACCESS NOT AUTHORIZED

FILE 'BIOTECHDS' ENTERED AT 12:25:25 ON 11 NOV 2009
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FILE 'WPIDS' ENTERED AT 12:25:25 ON 11 NOV 2009
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FILE 'WPINDEX' ACCESS NOT AUTHORIZED

=> s L6
L7          72 L6

=> dup rem L7
PROCESSING COMPLETED FOR L7
L8           22 DUP REM L7 (50 DUPLICATES REMOVED)

=> s L8 and py<2005
   5 FILES SEARCHED...
L9          16 L8 AND PY<2005

=> d L9 ibib abs 1-16

L9   ANSWER 1 OF 16  CAPLUS  COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER:        2004:1043363  CAPLUS <<LOGINID::20091111>>
DOCUMENT NUMBER:         142:253721
TITLE:                   Chloroquine causes lysosomal dysfunction in neural
```

retina and RPE: Implications for retinopathy
AUTHOR(S): Mahon, G. J.; Anderson, H. R.; Gardiner, T. A.; McFarlane, S.; Archer, D. B.; Stitt, A. W.
CORPORATE SOURCE: Eye Department, Institute of Clinical Science, Royal Victoria Hospital, Belfast, UK
SOURCE: Current Eye Research (2004), 28(4), 277-284
CODEN: CEYRDM; ISSN: 0271-3683
PUBLISHER: Taylor & Francis The Netherlands
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Chronic use of chloroquine has been shown to induce numerous pathophysiol. defects in the retina. This drug has the ability to alter pH of intracellular compartments and lysosomal function of the retinal pigment epithelium (RPE) and retinal neurons may constitute the basis of chloroquine retinopathy. The aim of the current study was to investigate pathogenic alterations in retinal cells continuously exposed to chloroquine using appropriate in vivo and in vitro models. Male hooded Lister rats were implanted with osmotic mini pumps which released chloroquine continuously over a period of seven days. The eyes were processed for electron microscopy and ultrastructural abnormalities determined in the neural retina and quantified using stereol. in the retinal pigment epithelium (RPE). RPE were also exposed to chloroquine in vitro and lysosomal pH changes were investigated using a pH sensitive probe. Degradative capacity was also analyzed using FITC labeled rod outer segments (ROS). Chloroquine-treated animals displayed several ultrastructural abnormalities including numerous membranous cytoplasmic bodies (MCBs) in retinal neurons. Cone photoreceptors displayed numerous MCBs although rods did not. The RPE of the treated groups all showed significantly higher nos. of lysosomal associated organelles (LAO) than the control group ($p < 0.001$). The in vitro expts. demonstrated chloroquine-mediated rises in lysosomal pH and an increase in lysosome/phagosome accumulation of ROS in the chloroquine treated group ($p < 0.01$). The current study demonstrates that chloroquine disrupts lysosomal function in retinal neurons and RPE. The evidence presented provides a clear pathogenic basis for the functional defects experienced by patients with chloroquine retinopathy.
OS.CITING REF COUNT: 10 THERE ARE 10 CAPLUS RECORDS THAT CITE THIS RECORD (10 CITINGS)
REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 2 OF 16 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2003:474355 CAPLUS <<LOGINID::20091111>>
DOCUMENT NUMBER: 139:286273
TITLE: Retinoic acid-induced Golgi apparatus disruption in F2000 fibroblasts: A model for enhanced intracellular retrograde transport
AUTHOR(S): Tzankov, Alexandar
CORPORATE SOURCE: Institute of Pathology, University of Innsbruck, Innsbruck, A-6020, Austria
SOURCE: Journal of Biochemistry and Molecular Biology ( 2003), 36(3), 265-268
CODEN: JBMBE5; ISSN: 1225-8687
PUBLISHER: Biochemical Society of the Republic of Korea
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Retinoic acid (RA) can transform the Golgi apparatus (GA) into a diffuse vacuolar aggregate and increase the toxicity of some immunotoxins that enter into cells by receptor-mediated endocytosis. An ultramorphol. study of the RA-induced GA disruption was performed on F2000 fibroblasts. Cultures were treated with 0.11 to 30 μM RA for 7-180 min. The

endocytosis of Limax flavus agglutinin-peroxidase conjugate (LFA), and the interactions between a phorbol ester (PMA) and RA concerning GA disruption, were examined Exposure to 0.33 μM RA for 20 min transformed the GA into vacuolar aggregate. These vacuoles were not involved in endocytosis since they remained unstained after endocytosis of LFA. However, the lysosomes were involved in endocytosis, as they were strongly stained. Therefore, a RA-induced shift towards lysosomal routing of the entered LFA was presumed. Exposure to PMA made cells resistant to the Golgi-disturbing effects of RA, indicating that protein kinase C plays an important role in this process.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)

REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 3 OF 16 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2001:60963 CAPLUS <<LOGINID::20091111>>

DOCUMENT NUMBER: 134:309191

TITLE: Does A2E, a retinoid component of lipofuscin and inhibitor of lysosomal degradative functions, directly affect the activity of lysosomal hydrolases?

AUTHOR(S): Bermann, Marion; Schutt, Florian; Holz, Frank G.; Kopitz, Jurgen

CORPORATE SOURCE: Department of Pathochemistry and General Neurochemistry, Im Neuenheimer Feld 220/221, Germany

SOURCE: Experimental Eye Research (2001), 72(2), 191-195
CODEN: EXERA6; ISSN: 0014-4835

PUBLISHER: Academic Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB A2E is a retinoid component of lipofuscin and inhibitor of lysosomal degradative functions. It has been suggested that inhibition of the lysosomal degradative function of retinal pigment epithelium (RPE) cells by accumulating A2E is a causative factor in the pathophysiol. of retinal diseases associated with excessive lipofuscin accumulations, including age-related macular degeneration (ARMD) and Stargardt's disease. Therefore, the authors investigated the possibility that A2E was a direct inhibitor of one or more lysosomal hydrolases from cultured RPE cells. The effects of 0.1, 1 and 10 μM A2E on the activities of these hydrolases were measured in specific enzyme assays. All major classes of lysosomal hydrolases were covered including proteases, lipidases, glycosidases, nucleases, sulfatases, and phosphatases. All enzyme activities were detectable in cultured RPE cells. The results show that A2E, even at 10 μM, did not cause inhibition of any of the enzymes tested. In conclusion, because all tested enzyme activities remained unaffected by A2E, it is unlikely that a direct inhibition of lysosomal enzymes can explain the pathophysiool. role of A2E in ARMD and related diseases. (c) 2001 Academic Press.

OS.CITING REF COUNT: 16 THERE ARE 16 CAPLUS RECORDS THAT CITE THIS RECORD (16 CITINGS)

REFERENCE COUNT: 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 4 OF 16 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1999:152085 CAPLUS <<LOGINID::20091111>>

DOCUMENT NUMBER: 130:323405

TITLE: Melanin granules of retinal pigment epithelium are connected with the lysosomal degradation pathway

AUTHOR(S): Schraermeyer, Ulrich; Peters, Swaantje; Thumann, Gabriele; Kociok, Norbert; Heimann, Klaus

CORPORATE SOURCE: Department of Vitreoretinal Surgery, University of Cologne, Cologne, 50931, Germany
SOURCE: Experimental Eye Research (1999), 68(2), 237-245
CODEN: EXERA6; ISSN: 0014-4835
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Melanosomes are closely related to lysosomes and lipofuscin granules. This paper indicates the potential involvement of lysosomal degradation processes in retinal pigment epithelial (RPE) cells. RPE cells cultured on Bruch's membrane and choroid were fed with indigestible latex beads. The RPE cells from this preparation were treated with chloroquine to investigate whether membrane swelling, typical for lysosomes under this condition, can be induced in melanosomes. To investigate the fate of indigestible material associated with rod outer segments (ROS), gold-labeled ROS were injected transsclerally into the subretinal space of Long Evans rats using a 32 gauge Hamilton syringe. The degradation of labeled ROS was observed after 5 and 12 days by electron microscopy. The following results were observed Latex particles fuse with the melanin granules of the RPE. Following chloroquine treatment, the membranes of melanin granules fused, and formed large clusters and vacuoles. Gold granules were detected inside both early stage melanosomes and mature melanin granules of the RPE cells 5 and 12 days following subretinal injection of the labeled ROS. Higher nos. of gold granules were predominantly found in immature melanosomes containing still melanofilaments and in small fused mature melanin granules. In conclusion the effect of chloroquine clearly demonstrates that the melanosomes posses active proton pumps which is typical for lysosomes. In RPE cells stressed by overload with rod outer segments or by ingestion of undegradable material (latex beads, gold particles), fusion of these phagosomes with melanosomes of different maturity is more a general rule than an exception. Therefore, melanosomes are connected to lysosomal pathways in RPE cells. (c) 1999 Academic Press.

OS.CITING REF COUNT: 31 THERE ARE 31 CAPLUS RECORDS THAT CITE THIS RECORD (31 CITINGS)
REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 5 OF 16 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1995:1005284 CAPLUS <<LOGINID::20091111>>
DOCUMENT NUMBER: 124:44915
ORIGINAL REFERENCE NO.: 124:8211a,8214a
TITLE: Lipidemic effect as a manifestation of chloroquine retinotoxicity
AUTHOR(S): Gaafar, K. M.; Abdel-Khalek, L. R.; El-Sayed, N. K.; Ramadan, G. A.
CORPORATE SOURCE: Dep. Zool., Cairo Univ., Giza, Egypt
SOURCE: Arzneimittel-Forschung (1995), 45(11), 1231-5
CODEN: ARZNAD; ISSN: 0004-4172
PUBLISHER: Cantor
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The effect of long-term treatment of chloroquine (CAS 54-05-7) (20 mg/kg body weight) on serum lipid components and its relation to the retinotoxic effect was studied in albino rats. Chloroquine was found to form lamellar lysosome-like structures within the photoreceptor layer, as well as the pigment epithelium and neuroretinal layers. Biochem., hypolipidemia in the serum was observed mainly due to the decrease in phospholipid portion. It was hypothesized that due to the inhibition of the degradation

process in the defective lysosomes, the retinal cells were denied the re-use of their own phospholipids, and thereby resort to their uptake from the serum.

OS.CITING REF COUNT: 4 THERE ARE 4 CAPLUS RECORDS THAT CITE THIS RECORD (4 CITINGS)

L9 ANSWER 6 OF 16 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1995:533430 CAPLUS <<LOGINID::20091111>>
DOCUMENT NUMBER: 123:6438
ORIGINAL REFERENCE NO.: 123:1347a,1350a
TITLE: Enzymic digestion increases permeability of the outer blood-retinal barrier for high-molecular-weight substances
AUTHOR(S): Prunte, Christian; Kain, Hermann L.
CORPORATE SOURCE: University Eye Clinic, Basel, CH-4056, Switz.
SOURCE: Graefe's Archive for Clinical and Experimental Ophthalmology (1995), 233(2), 101-11
CODEN: GACODL; ISSN: 0721-832X
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The purpose of the study was to investigate whether lysosomal enzymes can participate in damaging the outer blood-retinal barrier and to examine the role of glycosaminoglycans in maintaining the barrier function for high-mol.-weight substances. The ciliary artery was cannulated in freshly enucleated pig eyes. Perfusion was performed with buffer (controls), with heparinase (substrate: heparan sulfate), or with lysosomal enzymes freshly prepared from pig retinal pigment epithelium at 36°C, followed by perfusion with the tracer native ferritin (NF) or the marker cationized ferritin (CF). The eyes were examined by electron microscopy. In controls treated with buffer alone, NF was found in high concentration in the lumina of the choroidal capillaries; however, little NF was found in Bruch's membrane (BsM). The tracer did not penetrate to any extent beyond BsM. In eyes digested with heparinase or lysosomal enzymes, significantly higher nos. of tracer mols. were found in BsM. Furthermore, NF penetrated BsM and was apparent in the subretinal space and also inside retinal pigment epithelial cells, probably due to pinocytosis. The results indicate that heparan sulfate proteoglycan is important for the maintenance of the outer blood-retinal barrier and that lysosomal proteases may participate in damaging this barrier, causing increased permeability to high-mol.-weight substances.

L9 ANSWER 7 OF 16 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1980:194829 CAPLUS <<LOGINID::20091111>>
DOCUMENT NUMBER: 92:194829
ORIGINAL REFERENCE NO.: 92:31540h,31541a
TITLE: The sources of acid hydrolases for photoreceptor membrane degradation in a grapsid crab
AUTHOR(S): Blest, A. D.; Stowe, Sally; Price, D. G.
CORPORATE SOURCE: Dep. Neurobiol., Aust. Natl. Univ., Canberra, Australia
SOURCE: Cell & Tissue Research (1980), 205(2), 229-44
CODEN: CTSRCS; ISSN: 0302-766X
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Dawn photoreceptor breakdown in Leptograpsus variegatus was analyzed at the ultrastructural level. Coated vesicles derived from microvilli were assembled as multivesicular bodies (mvbs), which degraded to multilamellar bodies (mlbs) and were lysed. Cytochem. markers for hydrolases were a F--inhibited β-glycerophosphatase and a F--insensitive p-nitrophenyl phosphatase, with indistinguishable distributions when localized at pH

5.0. These enzymes were injected into the secondary lysosomes from 2 sources. First, immediately after dawn Golgi bodies were highly active, and differentiated a transtubular network, from which tubules and vesicles detached, and could be seen fusing with mvbs and mlbs. Saccules derived from the rough endoplasmic reticulum provided a 2nd source and were most often seen in association with late mlbs. Both kinds of primary lysosome rarely gave acid phosphatase-pos. responses when free in the cytosol, but did so as they made contact with their secondary lysosomal targets. Lipid droplets and lipofuscin bodies were interpreted as the residual products of breakdown. These results are discussed in relation to previous findings on photoreceptor membrane breakdown in a dinopid spider. Attention is drawn to the implied diversity of organization of lysosomal compartments in receptors which internalize membranes of similar composition

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)

L9 ANSWER 8 OF 16 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1980:175910 CAPLUS <<LOGINID::20091111>>
DOCUMENT NUMBER: 92:175910
ORIGINAL REFERENCE NO.: 92:28415a,28418a
TITLE: Degradation of rhodopsin by a lysosomal fraction of retinal pigment epithelium: Biochemical aspects of the visual process. XLI
AUTHOR(S): Regan, C. M.; De Grip, W. J.; Daemen, F. J. M.; Bonting, S. L.
CORPORATE SOURCE: Dep. Biochem., Univ. Nijmegen, Nijmegen, 6500 HB, Neth.
SOURCE: Experimental Eye Research (1980), 30(2), 183-91
CODEN: EXERA6; ISSN: 0014-4835
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The degradation of rhodopsin, present in a photoreceptor membrane preparation, by a lysosomal fraction of retinal pigment epithelium and by the enzyme cathepsin D was studied. The lysosomal fraction was obtained by subcellular fractionation of bovine pigment epithelium on a linear sucrose gradient. This fractionation procedure showed the presence of 2 populations of lysosomes in the tissue which are thought to represent phagosomes and autophagous lysosomes. Cathepsin D was purified from bovine spleen by affinity chromatog. The lysosomal fraction and cathepsin D degraded nonilluminated and illuminated rod outer segment membranes similarly. However, the proteolytic rates after illumination were somewhat higher than before illumination. During the initial phase of degradation predominantly other membrane proteins than rhodopsin were attacked, whereas the spectral integrity of rhodopsin was retained and no alteration in photolytic behavior was detected. Subsequently, rhodopsin was slowly but completely degraded with concomitant loss of 500 nm absorbance. Na dodecyl sulfate gel electrophoresis showed that in addition to the 37,000-dalton band, which represents intact rhodopsin, new bands of 33,000-34,000 and 25,000-28,000 daltons appeared during the proteolytic reaction. Thus, the retinal pigment epithelium appears both to degrade rhodopsin sequentially to a specific 25,000-28,000-dalton fragment, and to degrade rhodopsin and its fragments nonspecifically to small peptides.

OS.CITING REF COUNT: 13 THERE ARE 13 CAPLUS RECORDS THAT CITE THIS RECORD (13 CITINGS)

L9 ANSWER 9 OF 16 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1977:14866 CAPLUS <<LOGINID::20091111>>
DOCUMENT NUMBER: 86:14866

ORIGINAL REFERENCE NO.: 86:2423a,2426a
TITLE: Influence of defective circulation of the posterior ciliary arteries on acid hydrolases in the choroid and the retina
AUTHOR(S): Hara, Satoshi; Hayasaka, Seiji; Kikuchi, Tadasu; Mizuno, Katsuyoshi
CORPORATE SOURCE: Sch. Med., Tohoku Univ., Sendai, Japan
SOURCE: Japanese Journal of Ophthalmology (1976), 20(3), 353-60
CODEN: JJOPA7; ISSN: 0021-5155
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The influence of ischemia by obstruction of the posterior temporal ciliary arteries on the rabbit retina and choroid was studied using standard biochem. methods. After dissection of the arteries, the unsedimentable activities of acid phosphatase, β-glucuronidase, and cathepsin D from the retina and the choroid increased markedly, while the total activities of these 3 enzymes were either constant or only slightly increased. The retina, whose adjoining choroidal circulation was occluded exptl., showed patchy degeneration. The ischemia followed by occlusive circulation in choroidal vessels perhaps caused the lysosomal particle to disrupt in the retina, and the lysosomal enzymes like acid phosphatase, β-glucuronidase, and cathepsin D were released from the lysosome particle and entered the unsedimentable fraction. Therefore, an autolytic process of the retina and choroid took place. Pathogenesis of some retinal diseases was discussed based on these results.
OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

L9 ANSWER 10 OF 16 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1970:518275 CAPLUS <<LOGINID::20091111>>
DOCUMENT NUMBER: 73:118275
ORIGINAL REFERENCE NO.: 73:19257a,19260a
TITLE: Changes of lysosomal enzymes during hereditary degeneration and histogenesis of retina in mice. I. Acid phosphatase visualized by azo-dye and lead nitrate methods
AUTHOR(S): Sanyal, Somes
CORPORATE SOURCE: Dep. Anat., Med. Fac. Rotterdam, Rotterdam, Neth.
SOURCE: Histochemie (1970), 23, 207-19
CODEN: HICHAU; ISSN: 0018-2222
DOCUMENT TYPE: Journal
LANGUAGE: English
AB In the normal histogenesis of mouse retina, localized distribution of acid phosphatase pos. granules has been seen around the photoreceptor cell nuclei along the outer limiting membrane. These granules disappear during the development of the rod elements. Temporarily increased activity is also seen along the nuclei of the inner layer adjacent to and in the course of the development of the outer and the inner plexiform layers. Within the inner nuclear layer, the cells at the outer and inner rows develop localized acid phosphatase pos. granules which persist in the adult retina. Ganglion cells and the layer of nerve fibers show little change. In the pigment epithelium the enzyme gradually increases. In mice homozygous for the retinal degeneration gene, degenerating photoreceptor cell nuclei, characterized by perinuclear acid phosphatase staining, can be detected before morphol. signs of degeneration. Increased frequency of such nuclei and intensity of staining are recorded with the progress of degeneration. Enzyme activity in the photoreceptor cells, within the inner nuclear layer and in the degenerating photoreceptor cell nuclei, is demonstrable using naphthol substrates but not β-glycerophosphate. Pos. reaction with β-glycerophosphate

is obtained in these sites in the presence of dimethyl sulfoxide. Existence of differential permeability among the retinal lysosomes is tentatively suggested.

L9 ANSWER 11 OF 16 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1969:104119 CAPLUS <<LOGINID::20091111>>
DOCUMENT NUMBER: 70:104119
ORIGINAL REFERENCE NO.: 70:19427a,19430a
TITLE: Acute effects of high vitamin A (alcohol, aldehyde, and acid) doses on the stability of lysosomes in perfused rat liver
AUTHOR(S): Frimmer, Max; Gries, J.; Waldvogel, G.
CORPORATE SOURCE: Inst. Pharmakol. Toxikol., Justus-Liebig-Univ. Giessen, Giessen, Fed. Rep. Ger.
SOURCE: Internationale Zeitschrift fuer Vitaminforschung ( 1968), 38(5), 454-8
CODEN: IZVIAK; ISSN: 0020-9406
DOCUMENT TYPE: Journal
LANGUAGE: German
AB Addition of 2500, 5000, 7500, or 10,000 immunization units of vitamin A acid (retinoic acid), vitamin A alc. (retinol), or vitamin A aldehyde (retinal) to the rat liver perfusate induced the release of β-glucuronidase from the liver cells. At the high concns. of the vitamin in the whole liver, a toxic effect was exerted on the lysosomal and other cellular membranes. At the lower doses, retinoic acid, retinol, and retinal induced the release of approx. equal quantities of β-glucuronidase.

L9 ANSWER 12 OF 16 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:226033 BIOSIS <<LOGINID::20091111>>
DOCUMENT NUMBER: PREV200400221138
TITLE: Effects of lysosomal A2E on lipid degradation in RPE.
AUTHOR(S): Rodriguez-Boulan, Enrique [Reprint Author]; Lakkaraju, Aparna; Leung, Larry; Finnemann, Silvia
CORPORATE SOURCE: Weill Medical College, Cornell University, New York, NY, USA
adc2001@mail.med.cornell.edu
SOURCE: Graefe's Archive for Clinical and Experimental Ophthalmology, (January 2004) Vol. 242, No. 1, pp. 60. print.
Meeting Info.: First Workshop on Cell Transplantation in Age-Related Macular Degeneration. Cologne, Germany. September 11-14, 2003.
CODEN: GACODL. ISSN: 0721-832X.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 21 Apr 2004
Last Updated on STN: 21 Apr 2004

L9 ANSWER 13 OF 16 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 1999:188045 BIOSIS <<LOGINID::20091111>>
DOCUMENT NUMBER: PREV199900188045
TITLE: Inhibition of lysosomal degradative functions in RPE cells by a retinoid component of lipofuscin.
AUTHOR(S): Holz, Frank G. [Reprint author]; Schuett, Florian; Kopitz, Juergen; Eldred, Graig E.; Kruse, Friedrich E.; Voelcker, Hans E.; Cantz, Michael
CORPORATE SOURCE: Department of Ophthalmology, University of Heidelberg, Im Neuenheimer Feld 400, D-69120, Heidelberg, Germany

SOURCE: IOVS, (March, 1999) Vol. 40, No. 3, pp. 737-743. print.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 5 May 1999
Last Updated on STN: 5 May 1999

AB Purpose. To investigate the effect of the lipofuscin component N-retinylidene-N-retinylethanolamine (A2-E) on degradative functions of lysosomes in human retinal pigment epithelial (RPE) cells and to evaluate its mechanism of action. Methods. A2-E was coupled to low-density lipoprotein (LDL). Human RPE cell cultures were loaded with the A2-E/LDL complex, and controls were run with medium containing LDL alone. To determine whether A2-E accumulated in lysosomes, cells were fractionated in a Percoll gradient, and protein degradation was determined by metabolic labeling and measurement of the release of low-molecular-weight radioactivity. Lysosomal degradation was distinguished from nonlysosomal degradation by inclusion of NH4Cl in the medium. The metabolism of sulfated glycosaminoglycans was studied by radiosulfate incorporation in pulse-chase experiments. Intralysosomal pH was determined using a fluorescent lysosomotropic pH indicator. Results. A2-E accumulated almost exclusively in the lysosomal compartment. Lysosomal protein degradation was reduced in a dose-dependent fashion in A2-E-treated cells. The selectivity of A2-E on lysosomal function was demonstrated by its lack of effect on degradation of extralysosomal protein. Lysosomal glycosaminoglycan catabolism of RPE cells was also strongly inhibited by A2-E. Lysosomal pH was increased by A2-E. Conclusions. The findings indicate that accumulation of A2-E in RPE cells interferes with lysosomal functions as exemplified by its inhibitory effect on protein and glycosaminoglycan catabolic pathways. The quaternary amine character of the A2-E apparently causes a perturbation of the acidic intralysosomal milieu, resulting in diminished hydrolase action and consequent accumulation of undegraded material. Such mechanism could be operative in retinal diseases associated with excessive lipofuscin accumulation including age-related macular degeneration.

L9 ANSWER 14 OF 16 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 1998:242335 BIOSIS <<LOGINID::20091111>>
DOCUMENT NUMBER: PREV199800242335
TITLE: A lipofuscin compound (A2-E) inhibits lysosomal degradative function in human RPE-cells.
AUTHOR(S): Schutt, F. [Reprint author]; Holz, F. G. [Reprint author]; Kopitz, J.; Kruse, F. E. [Reprint author]; Voelcker, H. E. [Reprint author]
CORPORATE SOURCE: Univ. Heidelberg, Dep. Ophthalmol., Heidelberg, Germany
SOURCE: IOVS, (March 15, 1998) Vol. 39, No. 4, pp. S729. print.
Meeting Info.: Annual Meeting of the Association for Research in Vision and Ophthalmology. Fort Lauderdale, Florida, USA. May 10-15, 1998. Association for Research in Vision and Ophthalmology.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 4 Jun 1998
Last Updated on STN: 4 Jun 1998

L9 ANSWER 15 OF 16 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 1971:42172 BIOSIS <<LOGINID::20091111>>

DOCUMENT NUMBER: PREV197107042172; BR07:42172
TITLE: AN INVESTIGATION INTO THE STRUCTURAL INTEGRITY OF LYSOSOMES AND ITS EFFECT IN THE NORMAL AND DYSTROPHIC RAT RETINA.
AUTHOR(S): BURDEN E M; READING H W; YATES C M
SOURCE: Experimental Eye Research, (1971) Vol. 11, No. 1, pp. 140.
CODEN: EXERA6. ISSN: 0014-4835.
DOCUMENT TYPE: Article
FILE SEGMENT: BR
LANGUAGE: Unavailable

L9 ANSWER 16 OF 16 Elsevier Biobase COPYRIGHT 2009 Elsevier Science B.V. on STN
ACCESSION NUMBER: 1999056348 ESBIOBASE <<LOGINID::20091111>>
TITLE: Inhibition of lysosomal degradative functions in RPE cells by a retinoid component of lipofuscin
AUTHOR(S): Holz, Frank G.; Schutt, Florian; Kruse, Friedrich E.; Volcker, Hans E.; Kopitz, Jurgen; Cantz, Michael; Eldred, Graig E.
CORPORATE SOURCE: Holz, Frank G.; Schutt, Florian; Kruse, Friedrich E.; Volcker, Hans E. (Department of Ophthalmology, University of Heidelberg, Im Neuenheimer Feld 400, Heidelberg (DE)); Holz, Frank G. (Department of Ophthalmology, University of Heidelbergm, Im Neuenheimer Feld 400, D-69120 Heidelberg (DE)); Kopitz, Jurgen; Cantz, Michael (Inst. of Pathochemistry/Neurochem., University of Heidelberg, Heidelberg (DE)); Eldred, Graig E.
SOURCE: Investigative Ophthalmology and Visual Science (1999) Volume 40, Number 3, pp. 737-743, 41 refs.
CODEN: IOVSDA ISSN: 0146-0404
COUNTRY OF PUBLICATION: United States of America
DOCUMENT TYPE: Journal; Article
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 31 Jan 2009
Last updated on STN: 31 Jan 2009
AN 1999056348 ESBIOBASE <<LOGINID::20091111>>
AB PURPOSE. To investigate the effect of the lipofuscin component N-retinylidene-N-retinylethanolamine (A2-E) on degradative functions of lysosomes in human retinal pigment epithelial (RPE) cells and to evaluate its mechanism of action. METHODS. A2-E was coupled to low-density lipoprotein (LDL). Human RPE cell cultures were loaded with the A2-E/LDL complex, and controls were run with medium containing LDL alone. To determine whether A2- E accumulated in lysosomes, cells were fractionated in a Percoll gradient, and protein degradation was determined by metabolic labeling and measurement of the release of low-molecular-weight radioactivity. Lysosomal degradation was distinguished from nonlysosomal degradation by inclusion of NH 4 Cl in the medium. The metabolism of sulfated glycosaminoglycans was studied by radiosulfate incorporation in pulse-chase experiments. Intralysosomal pH was determined using a fluorescent lysosomotropic pH indicator. RESULTS. A2-E accumulated almost exclusively in the lysosomal compartment. Lysosomal protein degradation was reduced in a dose-dependent fashion in A2-E-treated cells. The selectivity of A2-E on lysosomal function was demonstrated by its lack of effect on degradation of extralysosomal protein. Lysosomal glycosaminoglycan catabolism of RPE cells was also strongly inhibited by A2- E. Lysosomal pH was increased by A2-E. CONCLUSIONS. The findings indicate that accumulation of A2-E in RPE cells interferes with lysosomal functions as exemplified by its

inhibitory effect on protein and glycosaminoglycan catabolic pathways. The quaternary amine character of the A2-E apparently causes a perturbation of the acidic intralysosomal milieu, resulting in diminished hydrolase action and consequent accumulation of undegraded material. Such mechanism could be operative in retinal diseases associated with excessive lipofuscin accumulation including age-related macular degeneration.

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